Seminars in Colon and Rectal Surgery 30 (2019) 100690

Contents lists available at ScienceDirect

Seminars in Colon and Rectal Surgery

journal homepage: www.elsevier.com/locate/yscrs

Acute colonic pseudo-obstruction (Ogilvie's syndrome)

Cristina R. Harnsberger, MD

University of Massachusetts Memorial Medical Center, Division of Colon and Rectal Surgery, 67 Belmont Street, Ste. 201, Worcester, MA 01605, United States

ARTICLE INFO

Keywords:
Acute colonic pseudo-obstruction
Ogilvie's syndrome
Large bowel obstruction

ABSTRACT

Acute colonic pseudo-obstruction (ACPO), otherwise known as Ogilvie's syndrome, is a rare condition characterized by signs and symptoms of a large bowel obstruction in the absence of a mechanical cause. It typically involves the right colon and cecum, but can affect the entire large and small bowel. The underlying pathophysiology is incompletely understood, but is thought to be related in part to a disturbance in the autonomic innervation of the distal colon. The precipitating factors leading to ACPO are many, but it is often found in critically ill or institutionalized patients, in the setting of trauma or surgery, and in conjunction with electrolyte derangements. Presenting symptoms are similar to those of a large bowel obstruction. A soft, distended, and tympanitic abdomen are classic early in the disease process. Signs of sepsis, significant right lower quadrant or diffuse abdominal tenderness signify colonic ischemia or impending perforation. Work-up should exclude mechanical causes of obstruction and other etiologies of abdominal pain with laboratory studies, plain films, and cross-sectional imaging. The goal of management is to decompress the colon and thereby avoid risks of ischemia and perforation. Medical management is directed at correction of underlying illness and electrolyte abnormalities, minimizing predisposing medications, and bowel rest. If there is no improvement clinically within 48 h or the cecal diameter approaches 12 cm, neostigmine can be used if there is no contraindication. Should this fail or the patient not be a suitable candidate, colonoscopic decompression is employed with or without temporary tube decompression. Surgery is performed for medically refractory cases, if the cecal diameter is >12 cm, or there are signs of colonic ischemia or perforation. Surgical options include cecostomy, a colon resection with anastomosis, or subtotal colectomy with ileostomy and Hartmann's pouch depending on the clinical status of the patient and intraoperative findings.

Introduction

Acute colonic pseudo-obstruction (ACPO) is defined as signs and symptoms of a mechanical large bowel obstruction with accompanying colonic dilation that occur in absence of an organic cause. Sir William Ogilvie first described the syndrome in 1948 in his manuscript titled, "Large-intestine Colic due to Sympathetic Deprivation. A new clinical syndrome."[1] In the discussion of the manuscript, he described the syndrome as we know it today: "The symptoms and signs of colonic obstruction in these two cases, occurring in the absence of any organic disease…".

A review of 351 additional cases was published between Ogilvie's original article and 1980,[2] and currently it is estimated that colonic pseudo-obstruction accounts for 100 per 100,000 inpatient admissions per year[3]; as such, it is considered rare. ACPO typically involves dilation of the colon, which is most marked in the cecum and right colon, although the entire colon and small bowel can be dilated in some cases. It is more common in men and in those greater than 60 years of age. Historically, the mortality rate of ACPO was 25 to 31%.[2]

Pathophysiology

The pathophysiology of ACPO is incompletely understood, but a few predominant theories exist. In his original publication, Ogilvie postulated an imbalance of sympathetic and parasympathetic innervations, which is still the most widely-accepted theory today.[1] He proposed that an interruption of sympathetic input allowed parasympathetic fibers originating from the second through fourth sacral nerve roots to act unopposed. This can lead to tonic contraction of the distal colonic segment and functional obstruction.[4] However, more recently it has been thought that it is actually sacral parasympathetic denervation (rather than sympathetic) that can cause an atonic distal colonic segment similar to an adynamic ileus.[2] Nevertheless, an autonomic disturbance could be the link between colonic distension and the disorders with which it is associated.

In ACPO, the cecum is the most common site of perforation as dictated by the law of LaPlace, which states that wall tension is

Abbreviations: ACPO, Acute colonic pseudo-obstruction (ACPO); CT, Computed tomography

E-mail address: charnsbe@sansumclinic.org

https://doi.org/10.1016/j.scrs.2019.100690
1043-1489/

proportionate to the pressure multiplied by the radius. Classic teaching is that the risk of perforation increases significantly when the cecal diameter is greater than 10–12 cm. Additionally, a longer duration of colonic dilation has been implicated. If persistent colonic dilation is present greater than 6 days, risk of perforation increases.[5] There is data to suggest the duration of dilation may be more important than cecal diameter.[6]

Etiology

The etiology of ACPO is likely multifactorial, and many risk factors have been identified. It occurs disproportionately in patients of advanced age who are institutionalized, or hospitalized for severe illness, trauma, or surgery.[5,7] Associated metabolic derangements are common and can reflect dehydration. Hypokalemia, hypocalcemia, hypomagnesemia, and hypophosphatemia are noted most frequently; hypothyroidism has been implicated as well.[8]

With regard to severe illness, patients with significant cardiovascular, respiratory, neurological, metabolic, or infectious comorbid diseases are known to be at risk. A systematic review of the etiology of ACPO detailed these risk factors, among which are cardiogenic shock, myocardial infarction, congestive heart failure, chronic obstructive pulmonary disease, stroke, dementia, Parkinson's disease, pneumonia, varicella-zoster, and major burns.[9] Medications that predispose or exacerbate ACPO are more common in elderly or institutionalized patients, such as anti-cholinergics, calcium channel blockers, alpha 2 agonists, antipsychotics, dopaminergics, and opioids.[8]

Traumatic injury and surgery are frequently implicated in cases of ACPO. With regard to non-operative trauma, fractures or spinal cord injury are common perpetrators. An early report found the most common associated operations were cesarean section and hip surgery.[7] Although a rare surgical complication, currently ACPO is most frequently seen following orthopedic intervention at a rate of approximately 0.7–1.3%.[10] When observed following surgery, symptoms typically present an average of five days postoperatively.[10] ACPO is also seen following cardiac surgery, solid organ transplantation, pelvic or gynecologic surgery, and procedures performed under epidural or spinal anesthesia.[9] The association with pelvic procedures and regional anesthesia lends credence to the theory that disruption of autonomic innervation to the distal colon plays a key role in the syndrome's pathophysiology.

Signs and symptoms

The signs and symptoms of ACPO are similar to other causes of mechanical large bowel obstruction, and thus are not specific. The hallmark of ACPO is abdominal distension, which can develop gradually over a period of 5–7 days or more rapidly over 24–48 hours. In association with abdominal distension, mild abdominal pain, nausea, vomiting, constipation/obstipation, or even diarrhea can occur. In cases of marked abdominal distension, dyspnea can result due to upward movement of the diaphragm.[11]

Upon examination, the patient's abdomen is typically soft, but distended and tympanitic. Mild tenderness can be present with a viable colon. However, fever, tachycardia, severe abdominal pain, or peritonitis can signify colonic ischemia or perforation. Laboratory values can be normal in uncomplicated cases, or reflect metabolic derangements as described above, particularly in the case of dehydration. If significant leukocytosis or lactic acidosis is present in the absence of concurrent infection, colonic ischemia should be suspected.

Differential diagnosis

It is critical to rule out causes of mechanical large bowel obstruction and inflammatory processes prior to concluding that the etiology of a patient's symptoms is ACPO. The differential diagnosis includes colorectal malignancy, incarcerated hernia, volvulus, constipation, mesenteric ischemia, colonic stenosis, diverticulitis, toxic megacolon, Clostridium difficile colitis, and Hirschsprung disease.

Work-up

The work-up of suspected ACPO involves a thorough history with attention to the time-course of the disease process and associated risk factors, in addition to a complete physical examination that includes a digital rectal examination. The history and examination alone may be sufficient to identify an alternate cause of large bowel obstruction.

Laboratory work should include a complete blood count to evaluate for leukocytosis that may reflect an underlying infectious cause or impending perforation, as the white blood cell count is typically normal in uncomplicated cases of ACPO. A basic metabolic panel should be obtained to detect electrolyte abnormalities, which are present in greater than 50% of cases,[8] and evidence of acute renal injury suggestive of dehydration. A liver panel with amylase/lipase is evaluated to rule out other causes of abdominal pain, and a thyroid stimulating hormone level to detect hypothyroidism. If the patient has diarrhea, stool studies and testing for Clostridium difficile should be performed.

Diagnostic imaging includes plain roentgenogram and cross sectional studies at minimum. Although abdominal roentgenogram is often inadequate to confirm the diagnosis, typical features associated with ACPO have been described, and can help differentiate from other etiologies that present with similar symptoms; plain films may demonstrate continuous gaseous dilatation, minimal fluid, preserved haustrations, thin well-defined septa, smooth inner colonic contour, and normal consistency of fecal contents (Fig. 1).[12] In contrast, one may see thumbprinting or significant intraluminal colonic fluid with abnormal haustrations and a large fecal burden in the case of toxic megacolon or mechanical obstruction.

Computed tomography (CT) scan is greatly helpful in ruling out a mechanical cause of large bowel obstruction, and is utilized routinely with a diagnosis of ACPO is suspected. There is often a gradual transition point near the splenic flexure without an associated underlying organic cause, and a normal haustral pattern. If the diagnosis remains unclear or the patient cannot undergo a CT scan, endoscopic

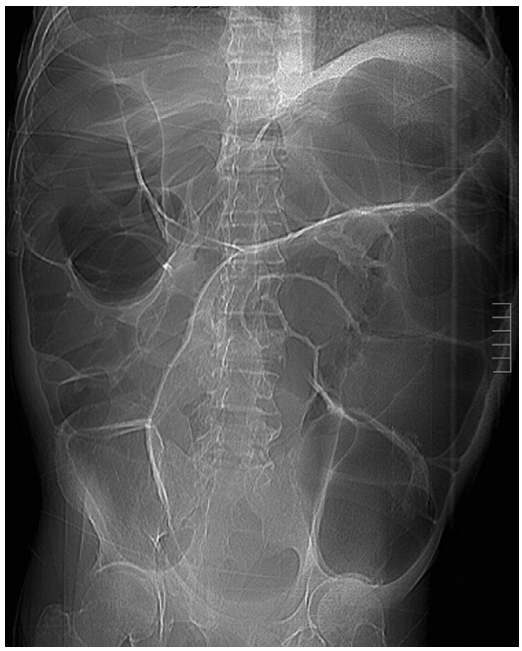

Fig. 1. Plain film in a patient with ACPO secondary to Tuberculosis. Courtesty of Samuel Eisenstein, M.D., University of California, San Diego.

evaluation or contrast enema can be used. These studies should be performed with caution so as not to traumatize an already tenuous colon. If there is concern for colonic ischemia or suggestion of peritonitis, they should be avoided so as not to increase intraluminal pressure and cause perforation. Despite the associated risks of contrast enema and diagnostic colonoscopy for suspected ACPO, they can provide potential therapeutic benefit, which will be discussed in the following section.

Management

Medical management

The goal of ACPO management is to decompress the colon to minimize risk of ischemia and perforation. Patients are typically admitted to a monitored unit, particularly if significant electrolyte abnormalities are present. Bowel rest is initiated, with nasogastric tube and rectal tube insertion to facilitate decompression.[13] Intravenous hydration is instituted and electrolyte derangements are corrected. Bed rest should be eliminated and the patient ambulated frequently unless contraindicated. If the precipitating cause is known, it is corrected and any exacerbating medications are minimized. The treatment team should perform serial abdominal examination to evaluate for change in distension and tenderness, so as not to miss early evidence of colonic ischemia. Serial laboratory studies are obtained to ensure correction of metabolic abnormalities and to detect early signs of colonic ischemia such as development of leukocytosis or lactic acidosis. Daily abdominal roentgenogram can be used to follow the extent and change of colonic dilation.

The most effective medication for treatment of ACPO is neostigmine, which is a peripheral-acting parasympathomimetic and reversible competitive inhibitor of acetylcholinesterase. It exerts is effect by increasing the amount of acetylcholine in the neuromuscular junction, indirectly stimulating the M2 and M3 muscarinic receptor subtypes in the muscularis mucosa and muscularis propria of the colon that produce smooth muscle contraction.[14] It should be considered if there is persistent colonic dilation after greater than 48 h of conservative management or cecal diameter greater than 12 cm without signs of ischemia, and is more effective than placebo.[15] Contraindications to its use include recent myocardial infarction, asthma, bradycardia, acidosis, peptic ulcer disease, and current beta blockade. Neostigmine is given intravenously in a 2 mg dose over 5 min with the patient under continuous cardiac monitoring.[15] Atropine should be readily available should bradycardia develop. A second dose can be given, and the dose can be reduced in the setting of bradycardia development or glycopyrrolate can be co-administered for prevention thereof. The onset of action of neostigmine is approximately 30 min, with a duration less than 2 h.[5] A meta-analysis demonstrated a single dose of neostigmine to be 89.2% effective in resolution of colonic distension compared to 14.7% in controls.[16] Abdominal pain was the most common side effect, occurring in 53% of patients, followed by sialorrhea in 31%, vomiting in 16%, and bradycardia in 6.3%. In a small randomized trial, patients who respond to neostigmine were given oral polyethylene glycol or placebo, and the former resulted in no recurrence of colonic dilation compared to 33% of patients administered a placebo.[17]

Procedural or surgical management

In patients who do not respond to medical management, endoscopic or surgical management is employed. Colonoscopic decompression should be performed with minimal insufflation using carbon dioxide to facilitate rapid reabsorption. Bowel preparation should not be attempted, as it can exacerbate colonic dilation. A flexible rectal tube can be placed over a guidewire into the right colon under fluoroscopic guidance, and remain in place to facilitate continued decompression thereafter.[5,18] Similar to its administration following successful decompression with neostigmine, oral polyethylene glycol can be given to prevent recurrent colonic dilation. There is some evidence to suggest administration of methylnaltrexone (a peripherally-acting opioid receptor antagonist) prior to colonoscopic decompression can improve durability of the colonic decompression.[19]

Surgery is indicated in patients with evidence of colonic ischemia, perforation, or in those who fail endoscopic decompression. If the colon is viable, tube cecostomy of cecostomy has success rates reported between 95–100%.[7,13,20] Although there is no comparative data between the two approaches, percutaneous tube cecostomy can be performed under endoscopic and fluoroscopic guidance on patients who are not surgical candidates.[5] In cases of colon ischemia or perforation, colon resection is performed with creation of an end ileostomy and Hartmann's pouch. Decision to perform an anastomosis with or without proximal diversion follows the general principles applicable to bowel surgery.[21,22]

Prognosis

In patients with ACPO who have no evidence of colonic ischemia, nonoperative management is successful within a few days in 70–90% of patients.[13] A series of 400 patients found evidence of ischemia or perforation in 0%, 7%, and 23% of patients with cecal diameters <12 cm, 12 to 14 cm, and >14 cm, respectively.[7] And although emergent presentations of ACPO are uncommon, ischemia or perforation occurs in 3–15% of cases with an associated mortality that has an upper range of nearly 50%.[2,7,13] In addition to colonic ischemia and perforation, risk factors for mortality in the setting of ACPO include advanced age, cecal diameter >14 cm, persistent colonic distension for >4 days, and requirement for operative intervention.[7,23]

Conclusion

Acute colonic pseudo-obstruction is a rare condition characterized by signs and symptoms of a large bowel obstruction in the absence of a mechanical cause, and may be due to an imbalance in the autonomic regulation of the distal colon. A high index of clinical suspicion in patients with known risk factors and diagnostic work-up to exclude organic causes of large bowel obstruction is required for diagnosis. Patients are managed nonoperatively if no signs of ischemia or perforation are present, and neostigmine is effective in the majority of cases. However, if patients fail medical management, colonoscopic decompression is attempted. In refractory cases and if there is evidence of colonic ischemia or perforation, surgical intervention is necessary with an associated high mortality rate.

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